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ABSTRACT

THE PURPOSE OF THIS STUDY WAS TO DETERMINE THE IMMEDIATE EFFECTS OF A CIGARETTE SMOKING ENVIRONMENT ON CHILDREN OF ELEMENTARY SCHOOL AGE. PHYSICAL EFFECTS WERE LOOKED FOR, AS WERE DIFFERENCES BETWEEN CHILDREN FROM SMOKING HOMES AND NON-SMOKING HOMES, AND MALE SUBJECTS AND FEMALE SUBJECTS. A TOTAL OF 103 CHILDREN WERE DIVIDED INTO TWO GROUPS, GROUP A PLACED IN BOTH A SMOKING AND NON-SMOKING ENVIRONMENT; AND GROUP B IN A SMOKING ENVIRONMENT ONLY. BASED ON THE RESULTS OF THE TESTS, THE FOLLOWING CONCLUSIONS WERE OFFERED: (1) CIGARETTE SMOKE WHICH ACCUMULATES IN POORLY VENTILATED ENCLOSURES INCREASED THE HEART RATE, BLOOD PRESSURE AND AMOUNT OF CARBON MONOXIDE IN NON-SMOKING ELEMENTARY SCHOOL CHILDREN; (2) THE SMOKING ENVIRONMENTS EFFECT UPON THE NON-SMOKER IN THE ENVIRONMENT IS SIMILAR TO THE CIGARETTE SMOKE'S EFFECT ON A SMOKER BUT ON A REDUCED SCALE; (3) NON-SMOKING ELEMENTARY SCHOOL AGE CHILDREN FROM NON-SMOKING HOMES REACT IN MUCH THE SAME MANNER TO A 30 MINUTE EXPOSURE TO A SMOKING ENVIRONMENT AS TO NON-SMOKING ELEMENTARY SCHOOL AGE CHILDREN FROM SMOKING HOMES. (AUTHOR/KJ)

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SOME IMMEDIATE EFFECTS OF A SMOKING

ENVIRONMENT ON CHILDREN OF

ELEMENTARY SCHOOL AGE

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Introduction

The identification of the smoking environment as a potential health hazard has begun to attract the attention of authorities in various fields. The National Clearinghouse for Smoking and Health has stated that evidence developed "in Switzerland and the United States tends to implicate certain cigarette gases as being more hazardous to health than tars and nicotine, which have been considered the major health villains of cigarettes." For investigative purposes cigarette smoke has been dichotomized into two phases: "mainstream" smoke and "sidestream" smoke. Mainstream smoke results when air is drawn through the burning tobacco into the oral cavity and sidestream smoke is that which enters the surrounding atmosphere. A majority of the previously reported studies relate to the characteristics of the smoker and non-smoker but very little information is available concerning the smoking environment or sidestream smoke. Evidence does show that sidestream smoke is oxidized more efficiently and could have lower carcinogenic hydrocarbons than mainstream smoke. Nevertheless, tar, nicotine and volatile irritant gases are present in the sidestream smoke.

The smoker usually bears the principle effects of smoking. However, when in an enclosed space with others present, he becomes a contributor to air pollution. In a poorly ventilated enclosure, such

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as a car or smoked-filled room, concentrations of irritant gases can easily reach several hundred parts per million. This exposes smokers and nonsmokers which are present to a toxic hazard. Several of these harmful gases have been identified. For example, carbon monoxide, nitrogen dioxide and hydrogen cyanide are potential mutagenic agents found in cigarette smoke. Scassellati et al. (9) found that the quantity of tar, nicotine and volatile irritant gases was constantly higher in sidestream smoke than the smoke inhaled by smokers. They also found that sidestream smoke from filtered cigarettes had a higher content of these mutagenic agents than sidestream smoke from cigarettes without a filter.

In the United States the first representative evidence against cigarette smoke as a household air pollutant was presented by Cameron (2, 3) in 1967 and 1968. He and his associates found that smokers' children are ill more frequently than nonsmokers' children. Most of the difference between the two groups were found to be in the respiratory disease category. Fullmer et al. (5) reported that certain lung diseases produce curshmann's type spirals in non-cellular bronchial secretions. In a study of 155 subjects in 1968, Fullmer and his associates found a significantly large number of these spirals in the sputum from respiratory tracts of apparently healthy male and female smokers. These spirals also appeared in sputum of one control group of nonsmokers who were exposed to tobacco smoke in their environment. This finding also indicates that second hand cigarette smoke may represent a distinct health hazard to nonsmokers in the environment.

However, the identification of cigarette smoke as an air pollutant

of potential health hazard to children raised in a smoking environment has been based on survey type studies alone. An exhaustive investigation of the literature revealed a lack of studies identifying the immediate effects of a cigarette smoking environment on children. Therefore it was the purpose of this study to determine the extent to which a cigarette smoking environment affects the nonsmoking child's heart rate, systolic and diastolic blood pressure.

Heart rate and blood pressure (systolic and diastolic) were selected as parameters because the literature revealed that smoking does cause profound changes upon the smoker in these areas. In a room where the smoke is allowed to become ambient, the immediate effects of the smoke on the children would be slightly reduced.

Methodology

Fifty-one children from the families of the faculty and staff at Texas A & M University were alternately exposed to a smoking and non-smoking environment to determine the effect of a smoking environment on the heart rate of elementary school age children. The special characteristics of the 51 subjects utilized to determine the different effects of these two environments on heart rate are shown in Table 1.

TABLE 1. SPECIAL CHARACTERISTICS OF THE SUBJECTS USED IN
DETERMINING THE EFFECT OF A SMOKING AND NONSMOKING
ENVIRONMENT ON HEART RATE

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Subjects	No. of N.S. Elem. School Children	Sex		Parental Smoking		
		M	F	0	1	2
6	7	3	4	2	2	3
7	4	2	2	1	2	1
8	8	4	4	5	1	2
9	11	10	1	3	4	4
10	4	1	3	2	2	0
11	7	2	5	2	3	2
12	4	3	1	2	2	0
13	6	3	3	3	3	0
9.8*		9.8*	9.9*	9.7*	10.6*	8.5*
Total Number	51	28	23	20	10	11

*Mean age

To determine the effect of a smoking and nonsmoking environment on elementary school age children's blood pressure, 40 of the original 51 subjects were selected. Eleven of the original 51 subjects had distorted systolic and diastolic readings due to a temporary malfunction of the infant blood pressure cuff and were not used. The special characteristic of the 40 subjects utilized to determine a smoking and nonsmoking environment on systolic and diastolic blood pressures are shown in Table 2.

TABLE 2. SPECIAL CHARACTERISTICS OF THE SUBJECTS USED IN
DETERMINING THE EFFECT OF A SMOKING AND NONSMOKING
ENVIRONMENT ON BLOOD PRESSURE

Age of Subjects	No. of N.S. Elem. School Children	Sex		Parental Smoking		
		M	F	0	1	2
6	3	2	1	1	0	2
7	3	1	2	1	1	1
8	5	2	3	3	0	2
9	9	8	1	3	2	4
10	4	1	3	2	2	0
11	6	1	5	2	2	2
12	4	3	1	3	1	0
13	4	1	3	1	1	2
9.8*		9.8*	9.9*	9.7*	10.6*	8.5*
Total Number	40	21	19	18	11	11

*Mean age

The Environmental Chamber

The Texas A & M University Industrial Engineering Department's Environmental Laboratory for Human Factors Research was used to simulate the controlled environment. The environmental chamber measured 12' X 7' X 7'. The chamber had three observation points constructed of standard thermophane with full sized one-way mirrors. An atmospheric environmental system was used to maintain the temperature between 70° and 75° F. Humidity was regulated by a large residential humidifier and was controlled within a range of 40 to 50 percent. Inside the chamber, three straight back chairs were placed six feet from the photographic screen. These chairs were occupied by the subjects during the experiment. Directly behind the subjects, two adjacent chairs were used by the adult smokers. Physiological data were obtained from the subjects by two E & M physiograph projector models type PMP 4A. Both machines were equipped with complete accessories which provide an electrocardiograph record of the heart rate. Two velco-touch and close adult cuffs and one velco-touch infant cuff with ten feet of tygon hose were used to measure the blood pressures of the subjects.

A movie film was used to aid the subjects in maintaining a quite, sedentary position during the collection of the physiological data. The audio-visual was a combination of two films concerning the harmful effects of smoking - "Time for Decision" and Huff the Puffless Dragon." The total viewing time was approximately 30 minutes.

Creating the Environments

The subjects were alternately tested once in a smoking environment

and once in a nonsmoking environment to determine the influence of a smoked-filled room on heart rate, systolic, and diastolic blood pressure.

The test-retest procedure is shown in Figure 1.

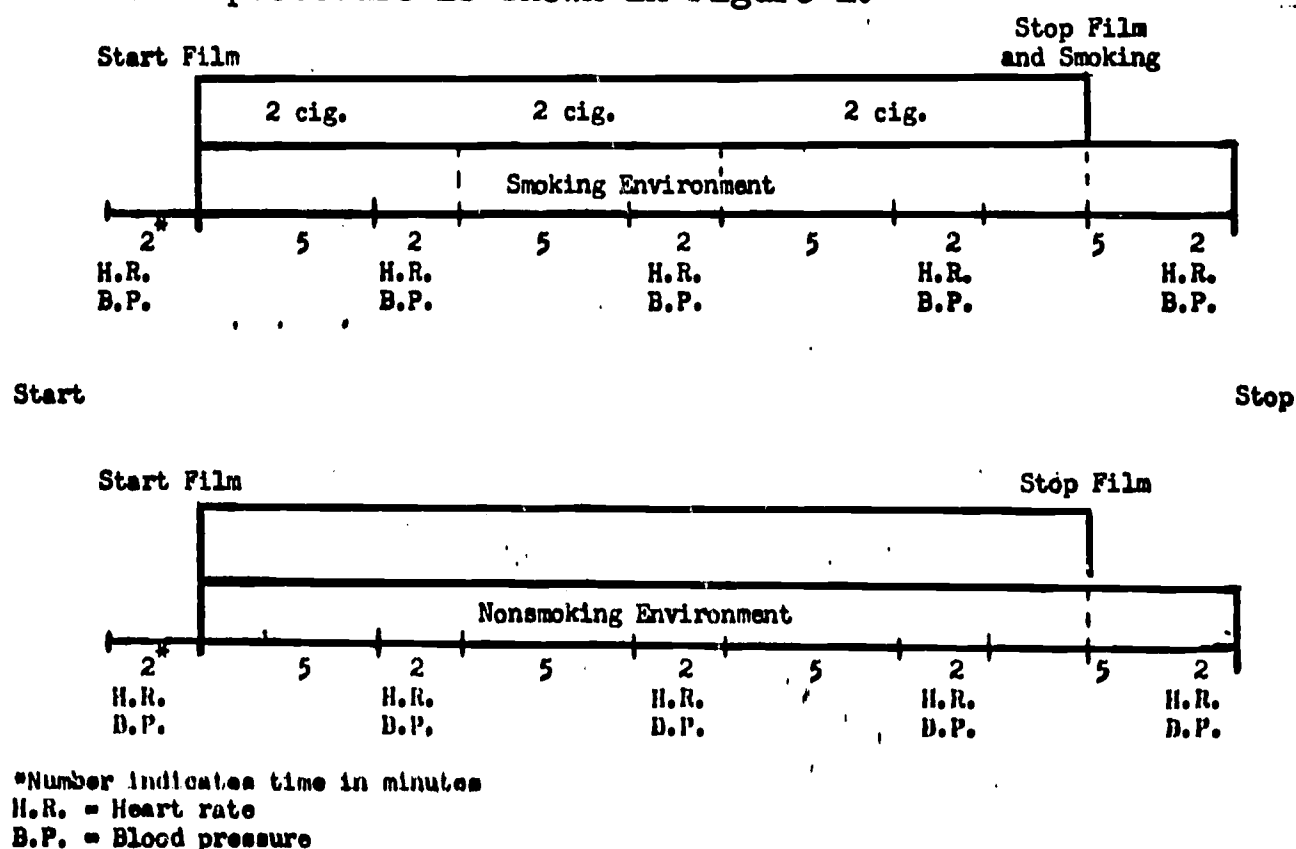


Figure 1. The test-retest procedure utilized in determining the effects of a smoking and nonsmoking environment on the 51 subjects.

The subjects were placed in the chamber and connected to the physiograph machines. After this was completed, one of the adult smokers turned off the automatic air conditioning system. All vents remained open, but in order to simulate a completely enclosed room, no fresh air was allowed to circulate through the room. The subjects were then given two to three minutes to adjust themselves, after which time the initial heart rates and blood pressures were recorded. The heart rates were monitored for two minutes while the blood pressures were taken three times. The average of each recording session was utilized in calculating beats per minute for heart rate and mm. of Hg. for blood pressure. At the termination of the first recording, the lights were dimmed and the audio-visual was started.

In creating the smoking environment, the film was the signal for the smokers to light one cigarette each. They were instructed to smoke at a rate that would burn the cigarettes to 1 - 1½ inch butts within seven minutes. Five minutes after the first recording, a second recording was transcribed. At the termination of the second recording, the two smokers lit a second cigarette each. Five minutes after the second recording, a third heart rate and blood pressure were taken, after which the smokers lit their third cigarette each. Five minutes later a fourth heart rate and blood pressure were recorded. Three minutes after the fourth recording, the film and the cigarette smoking were terminated. Two minutes later the heart rate and blood pressure were measured for the fifth and final time.

In simulating a nonsmoking environment, the same procedure was followed. However at the start of the film, the smokers pulled the cigarettes from the carton but did not light them.

Treatment of the Data

It was previously known that age influences heart rate and blood pressure, therefore the analysis of covariance, with age as the covariant, was used in determining the significance of the difference in the environmental effects on the subjects. These analyses were conducted with regard to the following classification: treatments, sex, parental smoking habits and the interaction of these classifications. All statistical tests were one-tailed and were conducted at the .05 level of confidence. All critical values were approximated by linear interpolation for the degrees of freedom listed in each table.

Analysis of the Data Recorded in Each Environment

The comparison of the effects of the two environments on the age-adjusted mean heart rates and blood pressures are presented in Figure 2.

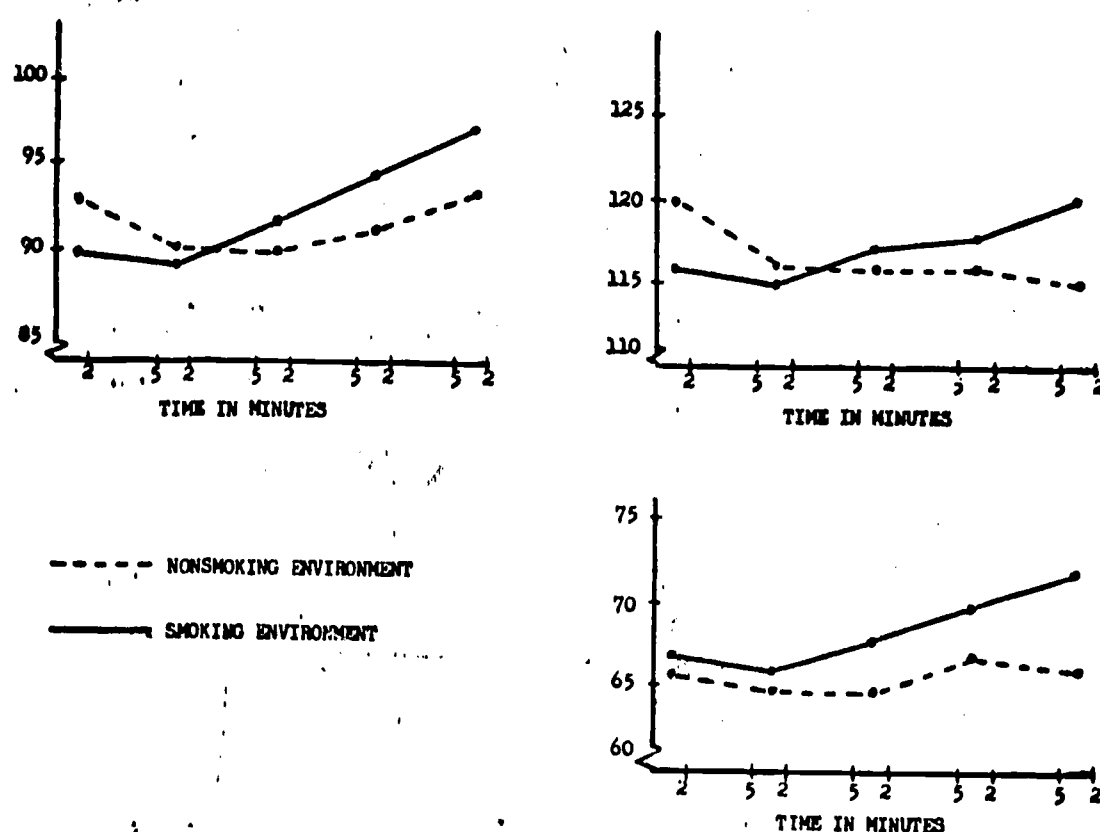


Figure 2. A COMPARISON OF THE AGE-ADJUSTED MEAN, HEART RATE, SYSTOLIC AND DIASTOLIC BLOOD PRESSURE, AT VARIOUS TIME INTERVALS IN A SMOKING AND NONSMOKING ENVIRONMENT.

The analyses of covariance, applied to the data, revealed that there was a significant difference in the treatment effect on the initial recordings of heart rate and systolic blood pressure. These data revealed that the age-adjusted mean heart rate and systolic blood pressure initially recorded in the nonsmoking environment were significantly greater than the age-adjusted mean heart rate and systolic pressure initially recorded in a smoking environment. The alternating research procedure used in the study was developed for the purpose of removing this variation. These phenomena were not anticipated, and in an endeavor to determine a reason for the differences, the investigator checked the differences in the mean heart rate, systolic and diastolic blood pressures

from the first to the second entry into the environmental chamber. The 25 subjects, who went from the nonsmoking to the smoking environment, had a mean heart rate decrease of 3 beats per minute, a mean systolic blood pressure decrease of 3 mm. Hg., and a mean diastolic blood pressure increase of 1 mm. Hg. The 26 subjects who went from the smoking environment to the nonsmoking environment had a mean heart rate increase of 4 beats per minute, a mean systolic blood pressure increase of 4 mm. Hg., and no mean difference in the diastolic blood pressure. These differences are illustrated in Table 3.

TABLE 3. DIFFERENCES IN THE MEAN HEART RATE, SYSTOLIC AND DIASTOLIC BLOOD PRESSURE OF THE SUBJECTS ON THE INITIAL RECORDINGS WITH RESPECT TO ORDER

Mean	Order						Total Diff.	F
	N.S. Env.	to S. Env.	Diff.	S. Env.	to N.S. Env.	Diff.		
Heart Rate	95	93	-3	86	90	4	7	2.99 ^a
Systolic Blood Pressure	121	118	-3	114	119	4	7 mm. Hg.	4.30 ^a
Diastolic Blood Pressure	66	67	1	67	67	0	1 mm. Hg.	.07

^aSignificant at the .05 level, one-tailed test.

$F_{.05}(1,90) = 2.77$.

$F_{.05}(1,68) = 2.785$.

N.S. = Nonsmoking
S. = Smoking

Although the heart rate, systolic and diastolic means were not adjusted for age, it does give some indication as to what occurred during the experiment. One explanation offered by this investigator was that the subjects were unaware of the type of environment they would experience. Therefore, the subjects that were exposed to the smoke first felt some discomfort and, thinking that the second trip to the environmental

chamber would be a similar unpleasant experience, manifested an anticipatory heart rate and systolic blood pressure increase. The graphs in Figure 2 indicate that 5 minutes following the initial recordings and after the film was started, both the age-adjusted mean heart rate and systolic blood pressure regressed to within 1 beat per minute and 1 mm. Hg. for both environments, respectfully. The initial diastolic blood pressure recording for each environment was not significantly different. The literature revealed that diastolic blood pressure was least affected by cigarette smoke, and since the environment does reduce the concentration of the smoke, the age-adjusted mean diastolic blood pressure showed only 1 mm. Hg. difference between the two environments on the initial recording.

The analysis further indicated that the smoking environment did not affect the blood pressure significantly until the fifth recording. This suggested that, by the time six cigarettes were smoked, the differences in the age-adjusted systolic and diastolic blood pressure due to the smoke were significantly greater than the age-adjusted mean systolic and diastolic blood pressure in the nonsmoking environment. However, the treatment effect on heart rate was not significantly different on the fifth recording. In studying the graphs presented in Figure 2, it was noted that the nonsmoking environment did produce a slight increase in heart rate, whereas the nonsmoking environment did not produce this same increase in blood pressure.

The analyses also indicated that there was a significant difference in the heart rates according to sex. This significant difference was noted at each of the five recordings. In checking the age-adjusted mean

heart rate it was found to be 88 beats per minute for the male subjects. However, the interaction of the treatments, by sex, was not significant at any one recording. This indicated that whatever effect the two environmental factors had on the group were the same for both the male and female subjects. The blood pressure readings, according to sex, were not significantly different nor were the treatments, according to sex, significantly different.

Although the analyses revealed that the order of placing the subjects in each environment were significantly different on the second and fifth recordings of heart rate, and the first and fifth recordings of systolic blood pressure, it was not consistently significant for all three variables of interest at each recording. The analyses also revealed that the parental smoking habits and the interaction of all other classifications were not significantly different. As was expected, the analyses revealed that the age-adjusted mean heart rate, systolic and diastolic blood pressure were significantly affected by the age of the subjects involved in the study.

Analysis of the Increases in Heart Rate, Systolic and Diastolic Blood Pressure

In order to analyze the difference in the increases of heart rate, systolic and diastolic blood pressure due to the total 30 minute exposure to a smoking and nonsmoking environment, linear slopes were calculated for each variable of interest in each environment. These slopes were based upon the age-adjusted mean heart rates, systolic and diastolic blood pressures recorded at each time interval on the subjects in each

environment. An analysis of covariance was then performed on the slopes to determine the significance of the differences between the two environmental effects.

Heart rate

The analysis indicated that the age-adjusted mean increase in heart rate in a 30 minute exposure to a smoking environment was significantly greater than the age-adjusted mean increase in heart rate in a 30 minutes exposure to a nonsmoking environment. The analysis further indicated that all other classifications and interactions on the variable of interest, heart rate, were not significant. A summary of this analysis is presented in Table 4.

TABLE 4. ANALYSIS OF COVARIANCE WITH RESPECT TO THE SLOPE
BASED ON THE AGE-ADJUSTED MEAN HEART RATES RECORDED
FROM THE FIRST THROUGH THE FIFTH TIME INTERVAL

Source of Variation	Degrees of Freedom	Adjusted Sums of Squares	Adjusted Mean Squares	F
Treatment	1	2579.87	2579.87	22.98 ^a
Sex	1	86.46	86.45	0.77
Order	1	62.11	62.11	0.55
Parental smoking	2	186.54	93.28	0.83
Treatment X sex	1	81.86	81.86	0.73
Treatment X parental smoking	2	503.93	251.97	2.24
Sex X parental smoking	2	301.16	150.58	1.34
Age	1	34.73	34.73	0.31
Error	90	10104.02	112.27	

^aSignificant at the .05 level, one-tailed test.
F₀₅ (1,90) = 2.77.

Systolic blood pressure

The analysis of covariance, applied to the data recorded on systolic blood pressure, revealed that the age-adjusted mean increase in systolic

blood pressure in a 30 minute exposure to a smoking environment was significantly greater than the age-adjusted mean increase in systolic blood pressure in a 30 minute exposure to a nonsmoking environment. The classifications, parental smoking habits and the interaction of sex by parental smoking habits, were significant for systolic blood pressure. However, this significant difference was not consistently noted for the heart rate or diastolic blood pressure. All other classifications and interactions of the environmental effects on systolic blood pressure were not significant. A summary of this analysis is presented in Table 5.

TABLE 5. ANALYSIS OF COVARIANCE WITH RESPECT TO THE SLOPE BASED ON THE AGE-ADJUSTED MEAN SYSTOLIC BLOOD PRESSURES RECORDED FROM THE FIRST THROUGH THE FIFTH TIME INTERVAL

Source of Variation	Degrees of Freedom	Adjusted Sums of Squares	Adjusted Mean Squares	F
Treatment	1	6710.07	6710.97	31.43 ^a
Sex	1	92.20	92.20	0.43
Order	1	313.92	313.92	1.47
Parental smoking	2	1225.54	612.77	2.87 ^a
Treatment X sex	1	231.43	231.43	1.08
Treatment X parental smoking	2	444.94	222.47	1.04
Sex X parental smoking	2	1826.14	913.07	4.28 ^a
Age	1	196.10	196.10	0.92
Error	68	14517.80	213.50	

^aSignificant at the .05 level, one-tailed test.
 $F_{.05}(1,68) = 2.785$.

Diastolic blood pressure

In comparing the difference in the diastolic blood pressure of elementary school age children due to the total 30 minute exposure in each environment, the analysis of covariance revealed that the increase in the age-adjusted mean diastolic blood pressure due to the 30 minute.

exposure to a smoking environment was significantly greater than the age-adjusted mean diastolic blood pressure due to a similar exposure in the nonsmoking environment. The analysis further indicated that the smoking environment had no significant influence on the age-adjusted mean diastolic blood pressure for any of the other classifications. A summary of this analysis is presented in Table 6.

TABLE 6. ANALYSIS OF COVARIANCE WITH RESPECT TO THE SLOPE BASED ON THE AGE-ADJUSTED MEAN DIASTOLIC BLOOD PRESSURES RECORDED FROM THE FIRST THROUGH THE FIFTH TIME INTERVAL

Source of Variation	Degrees of Freedom	Adjusted Sums of Squares	Adjusted Mean Squares	F
Treatment	1	2672.38	2672.38	14.46 ^a
Sex	1	289.67	289.67	1.57
Order	1	338.26	338.26	1.84
Parental smoking	2	113.82	56.91	.31
Treatment X sex	1	37.21	37.21	.20
Treatment X parental smoking	2	162.53	81.26	.44
Sex X parental smoking	2	141.43	70.72	.38
Age	1	.67	.67	.00
Error	68	1257.05	184.86	

^aSignificant at the .05 level, one-tailed test.
F₀₅ (1,68) = 2.785.

In an effort to eliminate the contamination, noted on the initial recordings, new slopes were calculated, based upon the age-adjusted mean heart rates, systolic and diastolic blood pressures recorded from the second through the fifth time interval. The analyses of the new slopes indicated that the increase in the age-adjusted mean heart rate, systolic and diastolic blood pressure, recorded in the smoking environment, were significantly greater than the age-adjusted mean heart rate, systolic and diastolic blood pressure recorded in the nonsmoking environment. The analyses further indicated that the classification, parental smoking

habits, was significant for heart rate and the classifications, sex, order and parental smoking habits, were significant for both systolic and diastolic blood pressure. However, the interactions of these classifications with the treatment effects were not significant for any of the three variables. A summary of these analyses appears in Tables 7 through 9.

TABLE 7. ANALYSIS OF COVARIANCE WITH RESPECT TO THE SLOPE
BASED ON THE AGE-ADJUSTED MEAN HEART RATES RECORDED
FROM THE SECOND THROUGH THE FIFTH TIME INTERVAL

Source of Variation	Degrees of Freedom	Adjusted Sums of Squares	Adjusted Mean Squares	F
Treatment	1	678.87	678.87	5.02 ^a
Sex	1	196.75	196.75	1.46
Order	1	61.74	61.74	.46
Parental smoking	2	2193.17	1096.58	8.11 ^a
Treatment X sex	1	18.29	18.29	.14
Treatment X parental smoking	2	445.11	222.58	1.65
Sex X parental smoking	2	644.90	322.45	2.38
Age	1	9.41	9.40	.07
Error	90	12168.53	135.20	

^aSignificant at the .05 level, one-tailed test.
 $F_{.05}(1,90) = 2.77$.

TABLE 8. ANALYSIS OF COVARIANCE WITH RESPECT TO THE SLOPE BASED
ON THE AGE-ADJUSTED MEAN SYSTOLIC BLOOD PRESSURES RECORDED
FROM THE SECOND THROUGH THE FIFTH TIME INTERVAL

Source of Variation	Degrees of Freedom	Adjusted Sums of Squares	Adjusted Mean Squares	F
Treatment	1	4975.63	4975.63	18.19 ^a
Sex	1	831.59	831.59	3.04 ^a
Order	1	1955.17	1955.17	7.15 ^a
Parental smoking	2	1776.46	888.23	3.25 ^a
Treatment X sex	1	21.02	21.02	.08
Treatment X parental smoking	2	898.03	449.02	1.64
Sex X parental smoking	2	2018.98	1009.49	3.69 ^a
Age	1	297.98	297.98	1.09
Error	68	18602.35	273.56	

^aSignificant at the .05 level, one-tailed test.
 $F_{.05}(1,68) = 2.785$.

TABLE 9. ANALYSIS OF COVARIANCE WITH RESPECT TO THE SLOPE BASED ON THE AGE-ADJUSTED MEAN DIASTOLIC BLOOD PRESSURES RECORDED FROM THE SECOND THROUGH THE FIFTH TIME INTERVAL

Source of Variation	Degrees of Freedom	Adjusted Sums of Squares	Adjusted Mean Squares	F
Treatment	1	3233.02	3233.02	12.51 ^a
Sex	1	205.86	205.86	.80
Order	1	499.11	499.11	1.93
Parental smoking	2	188.39	94.20	.36
Treatment X sex	1	.04	.04	.00
Treatment X parental smoking	2	359.92	179.96	.70
Sex X parental smoking	2	655.24	332.62	1.29
Age	1	1.79	1.79	.01
Error	68	17580.40	258.54	

^aSignificant at the .05 level, one-tailed test.
 $F_{.05}(1,68) = 2.785$.

Concluding Remarks

Based on the results obtained in this study, the following conclusions were offered: (1) cigarette smoke which is allowed to accumulate in a poorly ventilated enclosure significantly increases the non-smoking elementary school age children's heart rate, systolic and diastolic blood pressure, (2) the smoking environment's affect upon the non-smoker in the environment is similar to the cigarette smoke's affect upon the smoker but on a reduced scale, (3) nonsmoking elementary school age children from nonsmoking homes react in much the same manner to a 30-minute exposure to a cigarette smoking environment as do nonsmoking elementary school age children from smoking homes, and (4) both sexes seem to be affected by a 30 minute exposure to a cigarette smoking environment in the same manner.

REFERENCES

1. Abelson, Philip H. A damaging source of air pollution. Science 158:1, December 1967.
2. Cameron, Paul. The presence of pets and smoking as correlates of perceived disease. Journal of allergy 40:12-15, July 1967.
3. Cameron, Paul, and others. The health of smokers' and nonsmokers' children. Unpublished study, Wayne State University, January 1968.
4. Conroe, Julius H. The physiological effects of smoking. Physiology for physicians 2:1-6, January 1964.
5. Fullmer, Cyril D., and others. Sputum of chronic cigarette smokers. Rocky mountain medical journal 66:42-46, January 1969.
6. Furey, Sandy A., and others. The comparative effects on circulation of smoking tobacco and lettuce leaf cigarettes. Angiology 18:218-23, April 1967.
7. National clearinghouse for smoking and health (703) 557-6807:1-3, June 1968.
8. National interagency on smoking and health. Proceedings, world conference on smoking and health. Washington, D. C.: The Council, 1967.
9. Scassellati, Sforzolini G.; Pascasio, F.; and Savino, A. Catrame e nicotina nella porzione aspirata e nella porzione ambientale del fumo di vari tipi di sigarette. (Tar and nicotine contents both in inhaled smoke and in smoke dispersed in room-air by various cigarette brands.) Annali della sanità pubblica 27:1-16, Settembre-Ottobre 1966.
10. Scassellati, Sforzolini G.; and Savino, A. Valutazione di un indice rapido di contaminazione della fase gassosa del fumo. (Evaluation of rapid ambient contamination of cigarette smoke relative to the gas phase of the smoke.) Unpublished study, Istituto D'Igiene Dell' Università di Perugia, 1967.
11. Scott, Ronald B. Some medical aspects of tobacco smoking. British medical journal 1:671-5, March 1952.
12. U. S. Department of Health, Education and Welfare, Public Health Service. Smoking and health. The surgeon general's report. Washington, D. C.: Government printing office, 1964.
13. U. S. Department of Health, Education, and Welfare, Public Health Service. Health consequences of smoking. Washington, D. C.: Government printing office, 1967.